

Generalized Tetanus in an Adult Patient: A Case Report


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ABSTRACT

Tetanus is a neurological disorder caused by *Clostridium tetani*. Although vaccination has significantly reduced the incidence of tetanus worldwide, the occurrence of sporadic cases requires effective and timely treatment. In this study, we report the case of a 45-year-old man who had difficulty in swallowing and opening mouth. The patient had the history of a small puncture wound with a nail in his toes about 12 days ago.

Keywords: Tetanus, Toxin, Muscle spasms, *Clostridium tetani*, Immunization

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Introduction

Clostridium tetani is an obligate anaerobic, gram-positive, and spore-forming bacillus found globally in soil, intestinal tracts, and feces of different animals. In some cases, it may be present in the human gastrointestinal tract as a transient microflora. Tetanus toxin or tetanospasmin is the most important virulence factor of *C. tetani* that affects motor neurons causing tetanic convulsions. Tetanus is a life-threatening disease characterized by progressive muscle tension and spastic paralysis (1).

According to the Centers for Disease Control and Prevention (CDC), between 70 and 35 new cases of tetanus are reported annually most of which are related to the elderly population (over 60 years), suggesting a reduction in the protective effect of the primary vaccination (2). Drug users are another high-risk group. In developing countries, the mortality rate is high (about 28,000 deaths per 100,000 populations) (2). Two-thirds of global tetanus cases occur in South Africa, in which more than 40% are due to neonatal infections (3). In most cases, tetanus occurs as a result of a serious injury, such as puncture, laceration, or scratching. In some cases, no damage can be detected and chronic conditions such as skin lesions, abscesses, and gangrene may be involved (4). Moreover, the relationship between tetanus and burns, frostbite, middle ear infections, surgery, abortion, childbirth, drug abuse, and tattooing has been reported. In rare cases, the source of infection and mode of transmission

is unclear (5). *C. tetani* spores are very stable and resistant to environmental conditions, heat, and different antiseptics. Contamination of wounds with spores is common, but germination, growth, and production of the neurotoxin only occurs in wounds with low oxidation-reduction potential such as devitalized tissue, foreign bodies, or dirty wounds; this could facilitate the growth of anaerobic microbes. *C. tetani* is not able to induce inflammatory responses alone and the feature of infection remains benign at the portal of entry unless polymicrobial infection develops. In a contaminated wound, when vegetative cells lyse, toxin releases and spreads through the lymphatic system and blood, and then reaches the terminal of motor neurons. After the light chain of toxin enters the motor neuron travels to the neuron cell body at the spinal cord and brain stem in a retrograde manner. As a result, inhibitory neurotransmitters including gamma-aminobutyric acid (GABA) and glycine are not released from central inhibitory neurons. These neurotransmitters have an inhibitory effect on motor neurons and in the absence of this central inhibition, hyperactivity, uncontrolled muscle spasms and spastic paralysis can be developed. The binding of the toxin to the neuron is irreversible and recovery of the neural function can be achieved by new nerve terminals sprouting and new synapses formation (6, 7).

Case Report

A 45-year-old man with dysphasia and pain in the back of the head was admitted to the emergency department. The patient was unable to open his mouth. During the clinical examination, he was conscious and distressed, had hyperhidrosis, and uncontrollable laughing. At the time of hospital admission, the patient's symptoms were BP=175/90, HR=145, RR=30, BT=37, and SO₂=93% at room temperature. He had severe back pain when bending his legs and head and oral examination of the patient was not possible due to masseter muscle rigidity. He had no problem in upper limb function, and only a slight stiffness and tension was seen in the intercostal muscles. During lung auscultation, a reduction in lung sound intensity was detected.

Tachycardia and abdominal wall muscle rigidity were remarkable, but there was not any sensitivity, tenderness, and organomegaly. The tibialis anterior muscle was normal while the deep muscles of the posterior leg were tight. Plantar reflexes were normal and the presence of a scar between the first and second toes of the left foot was clear, though no active infection was found. The results of computerized tomography scan (CT scan) were normal, and the patient was subjected to tetanus treatment. Tracheal tube and antispastic drugs were used for opening airways and treatment of spasticity, respectively. Tetanus vaccine in conjunction with tetabulin were injected into different muscles and then administration of metronidazole and heparin prophylaxis was started. Concerning excessive sweating, high blood pressures, and tachycardia, metoral was given. Despite the onset of midazolam and diazepam medication, muscle spasms were rising, and sometimes severe generalized spasms happened. On day 3, with increasing masseter muscle spasm, the existing tracheal tube was replaced with a tracheostomy tube. Regarding the continuation of spasms, antispastic treatments were increased; but severe spasms of the Hamstring, back, neck, and intercostal muscles resulted in the addition of atracurium based on the recommendations of an anesthesiologist, and a ventilator was connected to the patient. In the following days, with

continuing hyperhidrosis, hypertension (>200/90), heart rate (>156), and a gradual and significant increase in CPK and CK-MB, the patient received captopril and aspirin in combination with metoral based on recommendations made by a cardiologist.

On day 8, when infusion dose of diazepam, midazolam, and atracurium reached to 12 mg/h, 20 mg/h, and 40 mg/h, respectively, intense and frequency of muscle spasm attacks were confined; but in certain circumstance, such as suction of discharges, he experienced high blood pressure and heart rate occasionally. The patient experienced a high fever (>38.8°C) from the second day of hospitalization. Therefore, gavage feeding was started using a nasogastric tube. Due to anemia and low MCV and MCH, a hemoglobin test was performed, the results of which showed that the amount of HbA₂ was about 4.6%. [Table 1](#) shows the laboratory test results on different days of hospitalization. During the hospitalization, two packed red blood cells were transfused due to hemoglobin decline, and there was no evidence of melena in the patient's stool examination. Atracurium was gradually reduced from day 9, then discontinued within one week.

By reducing muscle spasms, metronidazole completely discontinued on day 16 of hospitalization and the doses of diazepam and midazolam were gradually decreased so that on day 31, midazolam completely stopped and diazepam with a dose of 7 mg/h was continued. On day 40, the infusion of diazepam was stopped, and blood pressure, heart rate, and consciousness returned to normal conditions. Any obstruction and spasm of airways were checked by bronchoscopy and a tracheostomy tube was taken out on day 45. After physio consultation, physical therapy began. The patient was discharged from the ICU department with a good general condition on day 51. At that time, he had slight pain and swelling in the right knee, a significant reduction in weight, and needed support for walking.

Table 1. Blood monitoring of the patient with tetanus during hospitalization

	Day1	Day 4	Day7	Day10	Day12	Day21	Day31	Day48
WBC	9700	8300	8000		6400	6400	6700	6300
Hb	11.8	9.6	8.7		7.5	9.3	10	9.6
Hct	40.2	32.2	28.8		25.8	30.6	31.8	30.8
Plt	326000	211000	217000		296000	310000	342000	340000
BUN	64	43	20			22	105	12
Cr	1.1	1.1	0.8			0.7	0.8	0.8
Ca	8.8	8.7		9.1		8.9	9.4	9.6
P	3.2	2		3.2		5.7	4.2	4.4
Na	148	148	140	137		132	132	140

	Day1	Day 4	Day7	Day10	Day12	Day21	Day31	Day48
K	4.4	4.1	3.1	3.8		4.1	4.3	4.9
LDH		2385	972	1182	591			
CPK	1420	801	769	386	165			
CK-MB	39	88	64	42	13			

Discussion

C. tetani, as a causative agent of tetanus, is an anaerobic and spore-forming bacillus which is dispersed in the soil and environment. Neck stiffness, dysphagia, inability in opening the mouth, muscle rigidity, and painful spasms are the clinical features of tetanus disease. Since the eradication of *C. tetani* spores from the environment is not possible, active and passive immunization along with appropriate treatment of wounds and injuries are critical for tetanus prevention and management.

The diagnosis of tetanus is entirely based on clinical findings. Laboratory tests cannot be used to completely confirm or reject infection, and isolation of *C. tetani* from the wound has not enough sensitivity and specificity. However, gamma electromyography (EMG) may be useful (8-10). Several conditions, including alveolar abscesses, strychnine poisoning, drug-induced dystonia, and hypocalcemic tetany mimic tetanus, which must be differentiated from tetanus. Checking the precise vaccination history of individuals and possible traumas can be helpful in primary diagnosis and initiating appropriate treatment and preventive interventions (11, 12). The goal of the treatment is to eliminate the source of the toxin, neutralize the free toxin, control muscle spasms, monitor the patient's condition, and provide the necessary care, especially respiratory care. These considerations should be followed until the recovery of the patient. Toxin neutralization and eradication of *C. tetani* in infected people can be achieved by specific anti-toxin antibodies and antibiotics like penicillin or metronidazole, respectively. As the antibody titer decreases with age, vaccine and antibody should be injected after any burns, perforations, and crush injuries (13). Also, airway obstruction is one the most important complications of tetanus; therefore, tracheostomy or placing tracheal tube is recommended to open the airway urgently (14).

Conclusion

Although the incidence of tetanus in Iran with a regular schedule of vaccination program is rare, it is still a lethal disease and remains an important cause of death among preventable diseases by vaccination. Elderly people and neonates are at-risk patients and need more attention in this regard. Therefore, differential and appropriate treatment in line with booster immunization should be considered in high-

risk groups presenting muscular rigidity (15). In summary, in this case report, a 45-year-old man with oropharyngeal and generalized signs was successfully treated with antispastic agents, tetabulin, vaccination, and antibacterial therapy.

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Ethical considerations

Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.) have been completely observed by the authors.

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Conflict of Interest

Authors declared no conflict of interest.

References

1. Bennett JE, Dolin R, Blaser MJ. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases: 2-Volume Set: Elsevier Health Sciences; 2014.
2. Budd A, Blanton L, Grohskopf L, et al. Manual for the surveillance of vaccine-preventable diseases. Centers for Disease Control and Prevention. 2017.
3. Hammarlund E, Thomas A, Poore EA, et al. Durability of vaccine-induced immunity against tetanus and diphtheria toxins: a cross-sectional analysis. Clin Infect Dis. 2016;62(9):1111-8. [DOI:10.1093/cid/ciw066]
4. Hinfey P. Tetanus: Background pathophysiology, etiology. Medscape. 2017.

5. Malani PN. Harrison's principles of internal medicine. JAMA. 2012;308(17):1813-4. [DOI:10.1001/jama.308.17.1813-b]
6. Masuyer G, Conrad J, Stenmark P. The structure of the tetanus toxin reveals pH-mediated domain dynamics. EMBO Reports. 2017;18(8):1306-17. [DOI:10.15252/embr.201744198]
7. Yen LM, Thwaites CL. Tetanus. Lancet. 2019;393(10181):1657-68. [DOI:10.1016/S0140-6736(18)33131-3]
8. Simpson L. Botulinum neurotoxin and tetanus toxin: Elsevier; 2012.
9. Isono H, Miyagami T, Katayama K, et al. Tetanus in the elderly: the management of intensive care and prolonged hospitalization. Internal Med. 2016;55(22):3399-402. [DOI:10.2169/internalmedicine.55.7131]
10. Finkelstein P, Teisch L, Allen CJ, Ruiz G. Tetanus: A potential public health threat in times of disaster. Prehosp Disaster Med. 2017;32(3):339-42. [DOI:10.1017/S1049023X17000012]
11. Aqeel AY, Arishi HM, Ageel HI, Arishi NH. Epidemiological and clinical aspects of neonatal tetanus from a tertiary care hospital. Int J Pediatr Adolesc Med. 2017;4(2):71-4. [DOI:10.1016/j.ijpam.2016.10.001]
12. Rhinesmith E, Fu L. Tetanus disease, treatment, management. Pediatr Rev Am Acad Pediatr. 2018;39(8):430. [DOI:10.1542/pir.2017-0238]
13. Hamborsky J, Kroger A, Wolfe C. Epidemiology and prevention of vaccine-preventable diseases: the Pink Book: Course Textbook: Public Health Foundation; 2015.
14. McVicar J. Should we test for tetanus immunity in all emergency department patients with wounds? Emerg Med J. 2013;30(3):177-9. [DOI:10.1136/emmermed-2012-201193]
15. Govindaraj GM, Riyaz A. Current practice in the management of tetanus. Crit Care. 2014;18(3):145. [DOI:10.1186/cc13894]

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